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Acute Irreversible Hippocampal Toxic Encephalopathy Secondary to Cannabis Use: A Case Report

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Abstract **Case Report**

This case report presents a rare and severe complication of cannabis use in a 32-year-old male patient. The patient presented with status epilepticus and was found to have acute irreversible hippocampal toxic encephalopathy confirmed by MRI. Despite aggressive treatment with sedation and anti-epileptic therapy up to quad therapy, the patient's seizures were refractory to treatment. The patient initially regained consciousness, but later suffered a relapse of status epilepticus and respiratory distress. This case highlights the importance of considering acute irreversible hippocampal toxic encephalopathy as a potential complication of chronic cannabis use, and the need for healthcare professionals to be aware of this serious and potentially life-threatening condition.

Keywords: Hippocampal Encephalopathy, Cannabis, Status epilepticus, Refractory seizures, Intensive care, case report.

INTRODUCTION

The consumption of cannabis in adolescence or young adulthood is common. There are long-term consequences following excessive and/or chronic use, which are mostly neuropsychiatric or neurovascular, with damage to the hippocampus described and confirmed [1]. We report here the rare case of acute toxic hippocampal encephalopathy secondary to cannabis use.

PATIENT AND OBSERVATION

This is a 31-year-old man, a chronic smoker of 10 pack year with chronic cannabis use of unknown quantity, admitted to the emergency department for management of status epilepticus. On admission, the patient was found to be unconscious (post-critical GCS of 9) with no sensory-motor deficits or neck stiffness. The patient had a SpO2 of 90% in ambient air with slight tachypnea. The patient was hemodynamically stable with normal capillary glycemia, a temperature of 38.5°C, and initial investigations including a contrast-enhanced brain CT scan, toxicological and biological sampling, and a lumbar puncture ruled out meningitis, cerebral thrombophlebitis, and other metabolic, infectious, and vascular etiologies of status epilepticus. Toxicological tests (blood and urine) were exclusively positive for cannabis.

The patient's laboratory findings showed a leukocyte count of 10050/mm3, CRP of 13 mg/L, and a procalcitonin level of 0.08 ng/ml. The liver and kidney function tests as well as the electrolyte levels were normal, while the ECG showed a regular sinus rhythm. Since EEG was not available, an MRI was performed which showed bilateral hippocampal hyperintensities in T2 and FLAIR sequences, with mild diffusion restriction and swollen appearance of both hippocampi, without enhancement after gadolinium injection and no findings on the T2 sequence (Figure 1).

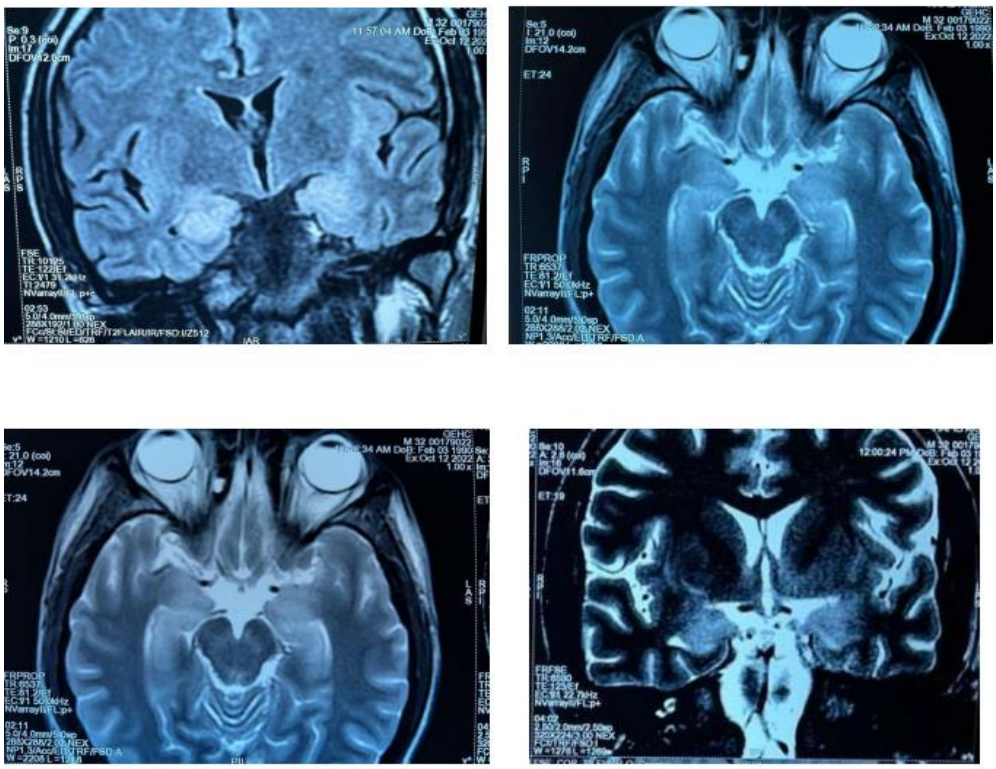

Figure 1: Bilateral Hippocampal Abnormalities on MRI: T2 and FLAIR Hyper intensities with Swollen Appearance and Mild Diffusion Restriction

DISCUSSION

Delta-9-tetrahydrocannabinol and cannabidiol are the active components of cannabis, which act on the CB1 cannabinoid receptors [2], highly expressed in the hippocampi [3]. Cannabis intoxication can cause psychiatric and cognitive disorders [4], and is considered one of the causes to investigate in cases of status epilepticus in young smokers, as the hippocampus is an epileptogenic zone. However, acute toxic hippocampal encephalopathy is a rare complication of chronic cannabis use, and we report a similar case in a cannabis consumer here.

We did not find any known etiology that could cause hippocampal lesions, such as ischemic stroke, herpes encephalitis, autoimmune encephalitis, limbic encephalitis, anoxic or hypoglycemic encephalopathy, or carbon monoxide poisoning [5].

These abnormalities, in addition to the status epilepticus, could correspond to the effects of two psychoactive components of cannabis, Δ-9-tetrahydrocannabinol (Δ-9-THC) and cannabidiol (CBD), which act on CB1 and CB2 receptors. The CB1 receptor is found in various sites of the body such as the heart, skeletal muscle, and kidneys [6, 7].

The Δ-9-THC is pro-convulsant [8], acute hippocampal encephalopathy can occur in cannabis users, however, there would be a genetic predisposition to this syndrome [9], in the literature cases of acute toxic hippocampal encephalopathy have been reported with more varied clinical features, namely: rhabdomyolysis, acute tubular necrosis of the kidneys, behavioral and memory disorders that can persist over time even after stabilization of the disease [3].

In terms of the management of our patient, intubation with sedation and anti-epileptic treatment up to quad therapy was initiated, as the seizures were refractory to treatment with seizures occurring after discontinuation of sedation. The initial course was favorable, with regaining of consciousness but without contact and with memory and behavioral disorders. Later during hospitalization, the patient had another epileptic status with generalized tonic-clonic seizures and associated respiratory distress.

Similar cases of toxic hippocampal encephalopathy manifesting as status epilepticus alone are extremely rare in the literature, and we report here a case that has never been described before.

CONCLUSION

This is a rare complication associated with cannabis, adding to the dangerous consequences of this drug. Healthcare professionals should be aware of the possibility of acute hippocampal encephalopathy in heavy cannabis users due to its serious and lasting effects.

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